Testimony To The United States House of Representatives
Committee On Commerce
Subcommittee On Oversight and Investigations and
Subcommittee on Health and Environment

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Mr. Chairman and Members of the Subcommittees:

Thank you for the opportunity to speak with you today regarding the particulate matter standard proposed by the Environmental Protection Agency (EPA). My name is Kevin Fennelly; I am an academic physician at the National Jewish Medical and Research Center in Denver, Colorado. I am board-certified in pulmonary medicine and in occupational-environmental medicine, and my time is evenly divided between patient care and clinical-epidemiological research. Most of the patients I see have asthma or chronic obstructive pulmonary disease (COPD), although I see patients with a wide spectrum of more unusual respiratory diseases. My research interests include the epidemiology of the health effects of particulate air pollution, so I am familiar with the scientific literature in this area.

I am testifying today as a concerned physician, scientist, and citizen. I support the EPA proposal, although a more stringent standard would provide additional public health benefits. I wish to emphasize three points. (1) Particulate air pollution causes human suffering, not just statistics. **(2)** There is biological plausibility to support the epidemiological findings of adverse health effects associated with particulate air pollution.

(3) The risk of adverse health effects due to particulate air pollution is comparable to other risks which our society has not found acceptable.

1. Particulate air pollution causes human suffering, not just statistics.

In discussing these issues with our local and state leaders, I realized that we physicians and scientists have not done an ideal job of communicating the meaning of recent scientific studies on particulate air pollution. The data have often been expressed in very abstract terms which are difficult to understand. My primary goal today is to try to bridge the gap between the scientific data and the clinical effects. I hope to prevent you from being numbed by all the numbers which you have undoubtedly seen, and to recall that behind all those statistics are people suffering from very real diseases.

As a physician specializing in lung diseases, I have seen patients who report worsening of their asthma symptoms on days of visible air pollution in Denver, Phoenix, Los Angeles, and the San Francisco Bay Area. These patients have told me of this association after a nonspecific inquiry about the triggers of their asthma symptoms, and they have not been aware of my research interest in air pollution. Colleagues have reported similar encounters. In Denver, our air pollution is predominated by particulate matter, so at least for our local patients, particulate air pollution is likely to contain the offending agent(s). Asthma is a common disease characterized by symptoms to multiple triggers, including respiratory infections, cold air, exercise, and other factors, including air pollution. Because of this, it is impossible in any one patient to quantify how much air pollution contributes to the disease. This speaks to the need for epidemiological studies

of groups of individuals to assess the relative contribution of factors such as air pollution.

Aside from asthmatics, another group susceptible to the effects of particulate air pollution are the elderly with heart or lung disease. Again since these diseases are so common, it is impossible for any one physician on any one day to notice changes in the pattern of illness or death which might be attributable to particulate air pollution. Even with the hundreds of deaths which occurred during the air pollution disaster in London in 1952, doctors did not appreciate the full magnitude of that public health disaster until the epidemiologic data were available.

Critics of the EPA proposal have also suggested that only asthmatics or elderly individuals with preterminal conditions are affected by particulate air pollution. This is also not true. In the air pollution disaster of 1952 in London, there were increased deaths among infants as well as in adults over the age of 45[1] Particulate air pollution has been associated with respiratory symptoms in children[2][3], as well as decreases in lung function in both asthmatic and nonasthmatic children[3] Especially concerning are recently reported associations of particulate air pollution and infant mortality in Czechoslovakia[4], Taiwan[5], and Rio de Janeiro[6] I understand that there is a paper in press reporting findings of infant mortality associated with particulate air pollution in the United States.

I have been disturbed by comments in the lay literature which have trivialized the occurrence of respiratory symptoms associated with air pollution. Breathing is our most basic function Without breath there is no life, and it should be understandable that

shortness of breath can be a distressing symptom. Allow me to suggest a simple exercise for those of you who may be fortunate enough to have escaped experiencing shortness of breath yourself or to have observed it in a family member. Simply take a drinking straw and breathe through it for several minutes, or better yet, try to walk about and climb some stairs. Then imagine feeling that way for hours or days. It is not a trivial discomfort.

The other disturbing suggestion I have heard is that patients with lung diseases should simply medicate themselves more to cope with air pollution. This is illogical and violates good medical practice. As an occupational pulmonologist, I engage considerable resources removing patients from exposures which may be causing or aggravating their asthma. In the case of urban air pollution, it is obviously impossible for patients to avoid breathing the air in their community Although inhaled bronchodilator medicines may be able to relieve symptoms temporarily, ongoing inhalation exposure will continue to aggravate the inflammation in the bronchial tubes which characterizes asthma and COPD. With more severe exacerbations, patients may have to use corticosteroid tablets or injections, which can have serious adverse effects if used repeatedly. Obviously infants and young children are not capable of using medications at will.

(1) There <u>is</u> biological plausibility to support the epidemiological findings of adverse health effects associated with particulate air pollution.

I will defer to Dr. Carl Shy's expertise in epidemiology to review the large numbers of studies which have found adverse health effects associated with particulate air

pollution, but I wish to offer a few observations. Critics of these studies have suggested that they are inconclusive or that they have been done by a small group of biased researchers from Harvard. In fact, there are now a large number of studies of various designs which have been done in various cities, countries, and climates, and by various investigators studying multiple outcomes: death rates, hospitalizations, emergency department visits, pulmonary function changes, asthma medication use, and symptoms. There has been a striking consistency in the findings of these studies. There have been a few studies which have not found similar results, but these have typically suffered from designs and methods which resulted in a lack of statistical power or the lack of a biologically plausible hypothesis.

Some critics of the EPA proposal have suggested that epidemiological studies are not valid science or use some sort of statistical sleight-of-hand. Advances in computing power and in statistical methods have improved the science modem epidemiology considerably, and this is similar to the advances due to improved technology in other fields. It is true that there have been epidemiologic studies of various suspected hazards which have resulted in associations which were later found to be spurious, In those cases the cause and effect relationship was readily dismissed after additional epidemiological and toxicological studies did not support the findiigs. However, this surely can not be an indictment against the field of epidemiology; similar processes occur in every field. In summary, it is highly unlikely that the epidemiological findings are due to chance or some other aberrations.

A common criticism expressed in the lay press has been the small magnitude of the

effects of the epidemiological studies. There have been references to the opinions of some scientists who only "accept" relative risks over 2 or 3 (or some other arbitrary number) in order to consider an association "significant". In fact, there is no consensus or "gold standard" in the scientific community for any criteria in this regard. Such criteria might be useful as a screen in assessing the value of one or even a few studies on a given subject. However, when there is a large body of literature which has demonstrated consistent results, as is the case regarding the health effects of particulate air pollution, we must accept the data as they are. The magnitude of the effects are indeed small at current levels of particulate air pollution, but they are consistent with the effects which occurred during severe air pollution episodes, such as in London, 1952. Indeed, this point satisfies another criteria for establishing a cause-and-effect relationship: a reasonable exposure-response relationship.

The impact on the public health is determined not only by the magnitude of the effect, but also by how many people are exposed and how frequently they are exposed. Highly toxic environmental hazards easily gain the attention of the media and the public. Conversely, exposure to urban air pollution is such a common experience that most people perceive very little risk. However, it can be as serious a public health risk albeit much more insidious, since there are large numbers of susceptible people frequently exposed to low concentrations of pollutants. Most of us were shocked at the accidental release of methyl isocyanate in Bhopal, India in 1984. There were at least 2000 deaths from that disaster [7], but the number of individuals dying from particulate air pollution each year clearly exceeds that number.

A common criticism of the EPA proposal for the particulate matter standard is that the epidemiological studies are not supported by biological plausibility. Although we still have much to learn this is not true. In the air pollution disaster in Donora, PA of 1948, there were symptoms in 88% of those with asthma, 77% of those with heart disease, and 79% of those with chronic bronchitis[8] There were 12 deaths in the Donora Borough during that week, which was six times the expected rate. Autopsies were performed on three of these patients. All three had evidence of capillary dilatation, edema, and hemorrhage in the lung with purulent bronchitis and bronchiolitis, which are inflammatory changes in the medium-to- large and small airways, respectively. All three of these patients had evidence of chronic cardiovascular disease. Similarly, in the killer fog of London in 1952, approximately 300 (60%) of over 500 autopsies demonstrated both heart and lung disease[l] Thus, the pathological data were consistent with the concurrent and more recent epidemiological findings of increased deaths due to heart and lung diseases.

Godleski and colleagues[9] recently presented preliminary findings of an inhalation toxicology study which was coherent with these pathological findings. They exposed rats with experimentally induced chronic bronchitis to concentrated urban air particulates. Those animals had a higher death rate (37%) than the controls (0%) as well as airway inflammation and marked constriction of the bronchial tubes.

Other animal studies have demonstrated lung inflammation and injury due to particulate matter, especially with very small particles described as “ultrafine”. [IO] There are a growing number of reports of investigations of the basic biological

mechanisms responsible for this inflammatory response, including free radical activity[11], prostaglandins[12], and endotoxin-induced activation of genes for cytokines, or chemical messengers[13] Another recent study[14] found that there is a marked increase in particle deposition in subjects with chronic obstructive lung disease, which may help explain the increased susceptibility of these individuals to the effects of particulate air pollution.

Although much more research is needed to elucidate the biological mechanisms causing the effects of particulate air pollutants, these early studies are already producing exciting results supporting the biological plausibility of the epidemiological findings. Some critics of the EPA proposal have called for more scientific certainty before taking action. As a pulmonologist, these arguments seem to echo the history of the science and public policy regarding cigarette smoking. Early epidemiological studies identified cigarette smoking as a risk factor for lung cancer and cardiovascular disease, but the strategy of the tobacco industry for years has been to repeatedly demand that more research is needed to confirm the hazards of cigarette smoking. Although we have learned a tremendous amount about the adverse health effects of cigarette smoking, we still do not know with absolute certainty exactly how smoking induces cancer and cardiovascular disease. However, few reasonable people now question the deleterious effects of cigarette smoking. Absolute certainty can be achieved only with complete convergence and consistency of all studies in all disciplines, including epidemiology, inhalation toxicology, dosimetry, and others. This has never happened, and it is highly unlikely that it will ever happen due to the nature of science as a human endeavor.

3. The risk of adverse health effects due to particulate air pollution is comparable to other risks which our society has not found acceptable.

Just as "absolute certainty" is impossible, there is no such thing as "zero risk." From a regulatory perspective, I can appreciate that this scientific literature is disturbing since there is no suggestion of a threshold concentration associated with these health effects. Therefore, the critical question becomes one of "acceptable risk" and of our societal values. Just as there is no gold standard for what constitutes a "significant" relative risk, there is no consensus as to what is an "acceptable risk" in our society. However, there are precedents suggesting at least a reasonable range. In the history of regulatory action in the U.S., the EPA and other agencies have often regulated hazards if the cancer risk were greater than 1 per 100,000[15] The Clean Air Act Amendments of 1990 mandated that the EPA regulate hazardous air pollutant emissions to reduce the risk to less than one in one million (See Sec. 112(f)(2)). In 1978, the Supreme Court suggested that an occupational risk of cancer due to benzene exposure of 100 per 100,000 warranted regulatory consideration[16] Thus, there is a range of lifetime risks for cancer from 1 per 1,000 to 1 per 1,000,000 which history suggests is not "acceptable" to our society. These data refer to risks for cancer, but it seems that an increased risk of death from heart or lung disease should be considered the same as an increased risk of death from cancer.

I would like to suggest an approach to help understand and communicate this issue: the use of incidence rates. EPA has typically performed risk assessments of carcinogenic hazards and expressed the risk in terms of deaths per 100,000 population. A similar

metric is used frequently in describing infectious disease risks, but I have not seen it used to describe risks from exposures to air pollution.

For example, to estimate the number of deaths attributable to PM,, in Denver, I assumed (1) a threshold effect of 30 mcg/m3 and (2) a 3.4% increase in respiratory deaths and a 1.4% increase in cardiac deaths for each 10 mcg/m3 increase in PM,, (average estimates suggested by Dockery and Pope[17]). Using the daily count of deaths and the daily PM,, concentrations for the city of Denver from 1990-92, I thus calculated 57 deaths, or an average of 19 cardiopulmonary deaths per year attributable to particulate air pollution. Since the population of the city of Denver in 1990 was 467,652, the annual crude cardiopulmonary mortality rate attributable to PM,, is 19/467,652, or 4 per 100,000. Since there were 1,745 cardiopulmonary deaths from 1990-92, 3.3% (57/1745) were attributable to PM,,. This conservative estimate is consistent with Lipfert's recent estimate that air pollution may account for 3-5% of deaths in affected urban areas; his estimate included lung cancer deaths as well. [18]

If the annual risk of death due to particulate air pollution is thus conservatively estimated at 4 per 100,000, then the cumulative risk over only 10 years of residence in this mildly polluted urban area would be 40 per 100,000. I also calculated similar risks for Philadelphia or Los Angeles using data provided in the EPA Staff Paper.[19] (See tables 1 and 2.) These estimates are substantantially larger, at 23 and 25 per 100,000 population per year, or 230 to 250 per 100,000 population over 10 years. Thus, the risk of acute cardiopulmonary death associated with particulate air pollution over a decade is similar to the "unacceptable" lifetime risk of cancer discussed above.

These risk estimates obviously do not include the many other nonfatal health effects of particulate air pollution, some of which are listed in tables 1 and 2. Although much emphasis has been placed on the studies of increased deaths associated with particulate air pollution, we know that mortality is only the "tip of the iceberg", i.e., that there are probably many more less serious adverse health effects if an exposure is able to produce death[20]

Such a discussion of quantitative risk estimates also does not include the qualitative aspects of risks associated with air pollution which the public has not found acceptable, such as exposures being involuntary, uncontrollable, and affecting children[21]

Summary

These issues are extremely complex, and in our struggles to be objective by providing quantitative data, it is easy to become numbed by the numbers. I see patients who have increased respiratory symptoms on days of high air pollution, and I suggest that we heed the medical maxim: "Listen to the patient." Behind the statistics are real people suffering with real symptoms. I congratulate the EPA in its review of the recent scientific literature and in recognizing the importance of $PM_{2.5}$. I have reviewed data supporting the biological plausibility of the numerous epidemiologic studies which have found multiple adverse health effects associated with particulate air pollution. I have also presented data indicating that Congress and the Supreme Court have historically not accepted health risks similar to those due to particulate air pollution. There are adequate data to support more stringent regulation of particulate air pollution, and the lack of "certainty" should not be an excuse for inaction. We could improve the public health by

implementing even more protective standards, such as those proposed by the American Lung Association. At minimum, I urge you to support the proposed changes in the particulate air pollution standard as proposed by the Environmental Protection Agency for $PM_{2.5}$, but with retention of the current PM,, standard to prevent any deterioration of air quality which could occur in western cities such as Denver under the EPA proposal.

Thank you for this opportunity to share my concerns.

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TABLE 1

Annual Risk of Health Effects Associated with Particulate Air Pollution: Philadelphia, PA 1992-93
(Events per 100,000 population at risk)
Calculated from Data in EPA 1996 **Staff Paper**: EPA-452 \ R-96-013

Health Effect	Population	Current PM10	Current PM2.5	Proposed 15 mcg/yr 50 mcg/d	Proposed 15 mcg/yr 25 mcg/d
Total Mortality (short-term exposure)	1,590,000	14	23	16	6
Total Mortality (long-term exposure)	1,590,000	N/A	58	25	0
Total Respiratory Hospital Admissions	1,590,000	N/A	16	11	5
Elderly Respiratory Hospital Admissions	241,000	104	N/A	N/A	N/A
Elderly Hospital Admissions for Chronic Obstructive Pulmonary Disease	241,000	50	N/A	N/A	N/A
Elderly Hospital Admissions for Prednisone	241,000	33	N/A	N/A	N/A
Elderly Hospital Admissions for Ischemic Heart Disease	241,000	33	29	21	8
Elderly Hospital Admissions for Congestive Heart Failure	241,000	46	42	29	12
Cases of Lower Respiratory Symptoms in Children	103,000	9,708	10,680	6,796	2,913
Days of Asthma Symptoms in Children	3,900	410,256	N/A	N/A	N/A
M.D. Diagnosed Bronchitis in Children	62,000	306	N/A	N/A	N/A

N/A = Data not available.

TABLE 2

Annual Risk of Health Effects Associated with Particulate Air Pollution: Los Angeles, CA 1995
(Events per 100,000 population at risk)
Calculated from Data in EPA 1996 Staff Paper: EPA-452 \ R-96-013

Health Effect	Population	Current PM10	Current PM2.5	Proposed 15 mcg/yr 50 mcg/d	Proposed 15 mcg/yr 25 mcg/d
Total Mortality (short-term exposure)	3,640,000	22	25	9	3
Total Mortality (long-term exposure)	3,640,000	N/A	80	N/A	N/A
Total Respiratory Hospital Admissions	3,640,000	N/A	33	11	4
Elderly Respiratory Hospital Admissions	322,000	332	N/A	N/A	N/A
Elderly Hospital Admissions for Chronic Obstructive Pulmonary Disease	322,000	137	N/A	N/A	N/A
Elderly Hospital Admissions for Pneumonia	322,000	130	N/A	N/A	N/A
Elderly Hospital Admissions for Ischemic Heart Disease	322,000	81	50	19	6
Cases of Respiratory Symptoms in Children	282,000	90	56	19	6
Days of Asthma Symptoms in Children	10,700	1,074,766	N/A	N/A	N/A
M.D. Diagnosed Bronchitis in Children	166,000	3,066	N/A	N/A	N/A
Days of Asthma Symptoms in Afr-Am Children	1,800	400,000	N/A	N/A	N/A

N/A = Data not available.

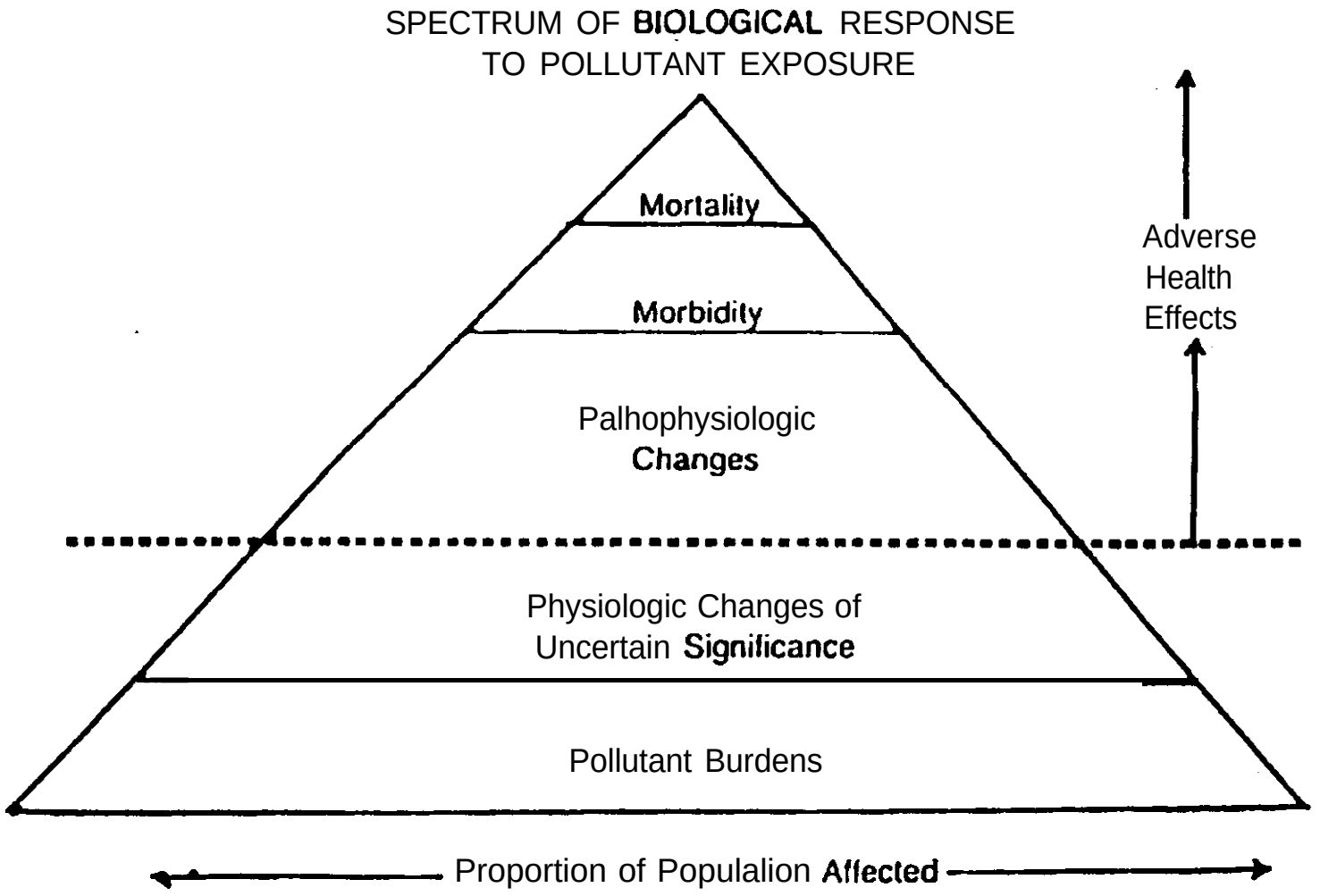

Fig. 1. Spectrum of biological response to pollutant exposure.

American Thoracic Society: Guidelines as to What Constitutes an Adverse Respiratory Health Effect, with Special Reference to Epidemiologic Studies of Air Pollution Am Rev Respir Dis 1985

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